EXHIBIT G

EXHIBIT I

UNITED STATES DISTRICT COURT
MIDDLE DISTRICT OF NORTH CAROLINA

JOAQUÍN CARCAÑO *et al.*, Plaintiffs, *v.* PATRICK MCCRORY *et al.*, Defendants	CASE NO. 1:16-CV-00236-TDS-JEP
UNITED STATES OF AMERICA, Plaintiff, *v.* STATE OF NORTH CAROLINA *et al.*, Defendants	CASE NO. 1:16-CV-00425-TDS-JEP

DECLARATION OF QUENTIN L. VAN METER, MD

1. I have been retained by counsel for Defendants as an expert in connection with the above-captioned litigation. I have actual knowledge of the matters stated in this declaration. My professional background, experience, and publications are detailed in my curriculum vitae, a true and accurate copy which is attached as Exhibit A to this declaration.

2. I received my B.A. in Science at the College of William and Mary, and my M.D. from the Medical College of Virginia, Virginia Commonwealth University.

3. I am currently a pediatric endocrinologist in private practice in Atlanta Georgia. I am the President of Van Meter Pediatric Endocrinology, P.C. I am on the clinical faculties of Emory University School of Medicine and Morehouse College of Medicine, in the role of adjunct Associate Professor of Pediatrics.

4. I am board certified in Pediatrics and Pediatric Endocrinology. I have been licensed to practice medicine in Georgia since 1991. I have been previously licensed to practice medicine in California, Louisiana, and Maryland.

5. I did my Pediatric Endocrine fellowship at Johns Hopkins Hospital from 1978-1980. The faculty present at that time had carried on the tradition of excellence established by Lawson Wilkins, M.D. Because of the reputation of the endocrine program as a center for exceptional care for children with disorders of sexual differentiation, I had well-above average exposure to such patients. As a Pediatric Fellow, I was also exposed to adults with Gender Identity Disorder, then called Trans-Sexuality, and received training from John Money, Ph.D., in his Psycho-hormonal Division.

6. I have maintained a continued interest in gender discordance since my fellowship years and have read extensively the literature in scientific peer-reviewed journals and have attended national and international pediatric endocrine conferences where this subject is presented and discussed. I am also familiar with the wide array of commentary on the subject.

7. My professional memberships include The Pediatric Endocrine Society, the Endocrine Society, the American Association of Clinical Endocrinologists where I hold a position on the Pediatric Scientific Committee, the American Diabetes Association, and I am a fellow of the American College of Pediatricians, currently serving on the Board of Directors as Vice

President. I am on the Board of Directors of Camp Kudzu, a non-profit organization which provides diabetes camp experience in Georgia.

8. My opinions expressed in this report are based upon my education, training, and experience in the subject matters discussed. The materials that I have relied upon are the same types of materials that other experts in my field rely upon when forming opinions. Specific sources upon which I rely in this declaration are footnoted.

9. Over my career, I have served as an expert witness in medical malpractice cases for both plaintiff and defense. I have testified at Georgia State Legislative Committee hearings. In the past four years, I have testified by deposition in *Harlen Schneider v. J. Enrique Lujan, M.D.et al.*, in the circuit court of the first judicial circuit of Okaloosa County, FL, Civil Division, on 7 Feb 2014; and in the case of plaintiff Kimora Gilmer, represented by attorneys at the Birmingham, AL, firm of Pittman Dutton on 22 May 2014.

10. My publications include a textbook chapter, case studies, and articles generated by clinical research studies. I serve on the speaker's bureau of major pharmaceutical companies.

11. I am being compensated at an hourly rate for actual time devoted, at the rate of $250 per hour. My compensation does not depend on the outcome of this litigation, the opinions I express, or the testimony I provide.

Sexual Differentiation in the Fetus

12. From the moment of conception, a fetus is determined to be either a male (XY), female (XX), or in rare cases, to have a combination of sex-determining chromosomes, many of which are not compatible with life, and some of which are the cause of identifiable clinical syndromes. The presence of a Y chromosome in the developing fetus directs the developing gonadal tissue to develop as a testicle. The absence of a functional Y chromosome allows the gonadal tissue to

develop as an ovary. Under the influence of the mother's placental hormones, the testicle will produce testosterone which directs the genital tissue to form a penis and a scrotum. Simultaneously, the testicle produces anti-Müllerian Hormone (AMH) which regresses development of the tissue that would otherwise develop into the uterus, fallopian tubes, and upper third of the vagina.

13. This combination of actions in early fetal development is responsible for what we subsequently see on fetal sonograms, and what we observe at birth as male or female genitalia. It is only when the genital structures are ambiguous in appearance that sex assignment is withheld until a thorough expert team evaluation has occurred.

14. For reasons most often occurring as random events, there are malfunctions of the normal differentiation. These aberrations of normal development are responsible for what we classify as Disorders of Sexual Differentiation (DSD) and they represent a very small fraction of the human population. The incidence of such circumstances occurs in 1:4500 to 1:5500 births.[1]

15. Sex is binary, male or female, and is determined by chromosomal complement and corresponding reproductive role. The exceedingly rare DSDs are all medically identifiable deviations from the human binary sexual binary norm. The 2006 consensus statement of the Intersex Society of North America and the 2015 revision of the Statement does not endorse DSD as a third sex.[2]

16. DSD outcomes range from appearance of female external genitalia in an XY male (complete androgen insensitivity syndrome) to appearance of male external genitalia in an XX female (severe congenital adrenal hyperplasia). As one would expect, there are variations of the

[1] Lee PA et al, Global Disorders of Sex Development Update since 2006: Perceptions, Approach and Care, 2016 Horm Res Paediatr.
[2] Lee PA et al, Consensus Statement on Management of Intersex Disorders, Pediatrics 2006; 118 e488-e500.

degree of hormonally driven changes that create ambiguous genital development that prevent assigning of a specific classification as either male or female at birth.

17. DSD patients are not "transgender"; they have an objective, physical, medically verifiable, physiologic condition. Transgender people generally do not have intersex conditions or any other verifiable physical anomaly. People who identify as "feeling like the opposite sex" or "somewhere in between" do not comprise a third sex. They remain biological men or biological women.

18. In some DSDs there exist more than one set of chromosomes. When there is a divergence of the appearance of the external genitalia from the chromosomally-determined sex due to the presence of both an ovarian and testicular cell lines in a patient simultaneously, the patient is classified as having ovo-testicular DSD (formerly termed a true hermaphrodite). When there is a disruption in the development of genital structures but there is solely testicular tissue present in the chromosomal male or solely ovarian tissue in the chromosomal female, the term 46 XY DSD or 46 XX DSD is used instead respectively (formerly termed male pseudohermaphrodite or female pseudohermaphrodite).

19. The decision to assign a sex of rearing is complex and is specific to the diagnosis. Patients with complete androgen insensitivity (CAIS) are XY DSD but are never reared as a male. Because testosterone never influences development, they become happy, functional female adults with infertility. Females with severe congenital adrenal hyperplasia (CAH) are XX DSD but are not reared as males despite the male appearance of the genitalia at birth. Although these girls may show a tendency for male play behaviors as children, they generally assume a female sexual identity.

20. Therapeutic interventions in the DSD individuals from infancy onward are aimed at what function can be expected from their disordered sexual anatomy in terms of function and fertility. Most often, the chromosomal sex aligns with the sex of rearing.

Gender Identity

21. "Gender" is a term that refers to the psychological and cultural characteristics associated with biological sex. It is a psychological concept and sociological term, not a biological one. The term gender possessed solely a linguistic meaning prior to the 1950s. This changed when sexologists of the 1950s and 1960s manipulated the term to conceptualize cross-dressing and transsexualism in their psychological practice.

22. "Gender identity" is a term coined by my former endocrine faculty member John Money in the 1970s and has come to refer to an individual's mental and emotional sense of being male or female. The norm is for individuals to have a gender identity that aligns with one's biological sex.

23. Gender discordance (formerly Gender Identity Disorder) is used to describe a psychological condition in which a person experiences marked incongruence between his experienced gender and the gender associated with his biological sex. He will often express the belief that he is the opposite sex.

24. Gender discordance occurs in 0.001% of biological females and in 0.0033% of biological males.[3] Exact numbers are hard to document since reporting is often anecdotal. Gender discordance is not considered a normal developmental variation.

25. "Gender Dysphoria" is a diagnostic term to describe the emotional distress caused by gender incongruity.[4]

[3] Seaborg E, About Face, Endocrine News 2014 (May) 16-19.
[4] American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 5th ed; 2013:451-459.

26. John Money played a prominent role in the early development of gender theory and transgenderism. He understood gender to be "the social performance indicative of an internal sexed identity."[5] He joined the Johns Hopkins faculty in 1951 specifically to have access to children diagnosed with DSD, hoping to prove his theory that gender was arbitrary and fluid.

27. Money experimented with DSD infants by assigning them to the opposite biological sex through surgical revision, counseling, and hormonal manipulation during puberty. His mode of operation was to have a theory and then experiment with patients to see how his theory worked. This kind of endeavor does not anticipate or prevent adverse outcomes and is the antithesis of ethical science. Money never submitted his research proposals for review; today, Institutional Review Boards (IRBs) serve to rigorously review proposed clinical research protocols to prevent all potential and real harm to patients.

28. Because of his experience with infants, Money initially garnered support from endocrine colleagues and surgical colleagues, and Johns Hopkins became a renowned center for care of patients with DSD in the 1970s, garnering referrals from around the world. Follow-up studies on these infants later showed, however, that altering their natal sexual identity via social intervention could lead to severe psychological harm. Clinical case reports of children with DSD have revealed that gender identity is indeed biologically influenced though not immune to environmental input.[6]

29. Meanwhile Money had expanded into the field of adult patients with persistent gender identity disorder. This very small group of patients chose voluntarily, as adults, to enter a very precise protocol which began with living socially as the opposite sex for a year, eventually receiving hormonal therapy to change their physical appearance to some extent. The final step

[5] Jeffeys, Sheila. Gender Hurts: A feminist analysis of the politics of transgendersim. Routledge. 2014 (p. 27).
[6] Whitehead, N. My Genes Made Me Do It. Chapter 5.

was surgical revision of the body structures that would otherwise be at odds with their desired gender. This small group of patients was followed for a number of years past their final surgical procedures and required continuous counseling. These patients expressed some degree of subjective satisfaction, but showed no objective improvement in overall wellbeing.[7]

30. The legacy of John Money fell into disrepute and the transsexual treatment program at Johns Hopkin was closed in the 1980s based on the lack of evidence that this protocol produced an effective cure.

Etiology of Gender Disorders

31. Transgender affirming professionals claim transgender individuals have a "feminized brain" trapped in a male body at birth and vice versa based upon various brain studies. Diffusion-weighted MRI scans have demonstrated that the pubertal testosterone surge in boys increases white matter volume. A study by Rametti and colleagues found that the white matter microstructure of the brains of female-to-male (FtM) transsexual adults, who had not begun testosterone treatment, more closely resembled that of men than that of women.[8] Other diffusion-weighted MRI studies have concluded that the white matter microstructure in both FtM and male-to-female (MtF) transsexuals falls halfway between that of genetic females and males.[9] These studies, however, are of limited clinical significance due to the small number of subjects and failure to account for neuroplasticity.

32. Neuroplasticity is the well-established phenomenon in which long-term behavior alters brain microstructure. For example, the MRI scans of experienced cab drivers in London are distinctly different from those of non-cab drivers, and the changes noted are dependent on the

[7] Meyer J.K. and Reter D. Sex Reassignment Follow-up . Arch. Gen. Psychiatry 36:1010-1015, 1979.
[8] Rametti G, Carrillo B, Gomez-Gil E, et al. White matter microstructure in female to male transsexuals before cross-sex hormonal treatment. A diffusion tensor imaging study. *J Psychiatr Res* 2011;45:199-204.
[9] Kranz GS, Hahn A, Kaufmann U, et al. White matter microstructure in transsexuals and controls investigated b diffusion tensor imaging. *J Neurosci* 2014;34(46):15466-15475.

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years of experience.[10] There is no evidence that people are born with brain microstructures that are forever unalterable, but there is significant evidence that experience changes brain microstructure.[11, 12] Therefore, any transgender brain differences would more likely be the result of transgender behavior than its cause.

33. Furthermore, infants' brains are imprinted prenatally by their own endogenous sex hormones, which are secreted from their gonads beginning at approximately eight weeks' gestation.[13, 14, 15] There are no published studies documenting MRI-verified differences in the brains of gender-disordered children or adolescents. The DSD guidelines also specifically state that current MRI technology cannot be used to identify those patients who should be raised as males or raised as females.[16]

34. Behavior geneticists have known for decades that while genes and hormones influence behavior, they do not hard-wire a person to think, feel, or behave in a particular way. The science of epigenetics has established that genes are not analogous to rigid "blueprints" for behavior. Rather, humans "develop traits through the dynamic process of gene-environment interaction. ... [genes alone] don't determine who we are."[17]

35. Regarding transgenderism, twin studies of adults prove definitively that prenatal genetic and hormone influence is minimal. The largest twin study of transgender adults found that only

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[10] Maguire EA et al, Navigation-related structural change in the hippocampi of taxi drivers, *PNAS* 2000;97:4398-4403.
[11] Gu J, Kanai R. What contributes to individual differences in brain structure? *Front Hum Neurosci* 2014;8:262.
[12] Sale A, Berardi N, Maffei L, Environment and Brain Plasticity: Towards an Endogenous Pharmacotherapy, *Physiol Rev* 2014; 94: 189 –234.
[13] Reyes FI, Winter JS, Faiman C. Studies on human sexual development fetal gonadal and adrenal sex steroids. *J Clin Endocrinol Metab* 1973; 37(1):74-78.
[14] Lombardo M. Fetal testosterone influences sexually dimorphic gray matter in the human brain. *J Neurosci* 2012; 32:674-680.
[15] Campano A. [ed]. Geneva Foundation for Medical Education and Research. *Human Sexual Differentiation*; 2016. Available at: www.gfmer.ch/Books/Reproductive_health/Human_sexual_differentiation.html. Accessed May 11, 2016.
[16] Lee PA et al, Consensus Statement on Management of Intersex Disorders, Pediatrics 2006; 118 e488-e500.
[17] Shenk, D. *The Genius in All of Us: Why everything you've been told about genetics, talent, and IQ is wrong*. (2010) New York, NY: Doubleday; p. 18.

20 percent of identical twins were both transgender-identified.[18] Since identical twins contain 100 percent of the same DNA from conception and develop in exactly the same prenatal environment exposed to the same prenatal hormones, if genes and/or prenatal hormones contributed to a significant degree to transgenderism, the concordance rates would be close to 100 percent. Instead, 80 percent of identical twin pairs were discordant. This would indicate that at least 80 percent of what contributes to transgenderism as an adult in one co-twin consists of one or more non-shared post-natal experiences including but not limited to non-shared family experiences.

36. These findings also mean that persistent GD is due predominately to the impact of non-shared environmental influences. These studies provide compelling evidence that discordant gender is not hard-wired genetically.

Gender Dysphoria vs. Gender Identity Disorder

37. Up until the recent revision of the DMS-IV criteria, the American Psychological Association (APA) held that Gender Identity Disorder (GID) was the mental disorder described as a discordance between the natal sex and the gender identity of the patient.

38. Dr. Kenneth Zucker, who is a highly respected clinician and researcher from Toronto carried on evaluation and treatment of GID patients for forty years. His works, widely published, found that the vast majority of boys and girls with GID identify with their biological sex by the time they emerge from puberty to adulthood, through either watchful waiting or family and individual counseling.[19] His results were mirrored in studies from Europe.[20, 21]

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[18] Diamond, M. "Transsexuality Among Twins: identity concordance, transition, rearing, and orientation." *International Journal of Transgenderism*, *14*(1), 24–38.
[19] Zucker KJ, Gender Identity Disorder, in Rutter M, Taylor EA, editors. *Child and Adolescent psychiatry*, 4th ed, Malden Mass: Blackwell, 2006: 737-753.
[20] Wallien MS, Cohen-Kettenis PT. Psychosexual outcome of gender-dysphoric children. *J AM Academy Child Adolescent Psychiatry* 2008; 47:1413-1423.

39. When the DMS-V revision of the diagnosis of GID was proposed by the APA committee responsible for revision, Dr. Zucker strongly opposed the change to the term Gender Dysphoria, which purposefully removed gender discordance as a mental disorder apart from the presence of significant emotional distress. With this revision, Gender Dysphoria describes the mental anguish which is experienced by the gender discordant patient.

40. The theory that societal rejection is the root cause of Gender dysphoria was validly questioned by a study from Sweden which showed that the dysphoria was not eliminated by hormones and sex reassignment surgery even with widespread societal acceptance.[22]

Treatment of Gender Dysphoria

41. The treatment of the child and adolescent with gender discordance and accompanying gender dysphoria should include an in-depth evaluation of the child and family dynamics. This provides a basis on which to proceed with psychologic therapy. The entire biologic and social family should be involved in psychological therapy designed to assist the patient, if at all possible, to align gender identity with natal sex. Psychological support by competent counselors with an intent of resolving the gender conflict should be provided as long as the patient continues to suffer emotionally. Given the high degree of eventual desistance of gender discordance/dysphoria by the end of puberty, it would be ethical and logical to counsel the patient and family to rear the child in conformity with natal sex.

42. There should be no interruption of natural puberty. Natural pubertal maturation in accordance with one's natal sex is not a disease. It is designed to carry a malleable, immature individual forward to be a healthy adult capable of conceiving their own progeny. It affects

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[21] Schechner T. Gender Identity Disorder: A Literature Review from a Developmental Perspective**.** *Isr J Psychiatry Related Sci* 2010; 47:42-48.

[22] Dhejne, Cecilia et al. Long-term Follow- up of transsexual Persons Undergoing Sex Reassignment Surgery: Cohort Study in Sweden *PLoS One* February 2011 Vol 6 Issue 2, e16885

physical changes, some of them painful, unique to the natal sex to reflect the laws of nature. Interruption of puberty has been reserved for children who begin puberty at an age much younger than normal in an effort to preserve final height potential and avoid the social consequences of precocious maturation. There are a number of physical changes that are a consequence of normally-timed puberty which could be classified as disadvantageous: changes in body proportions can alter success with dance and gymnastics; acne can be severe and disfiguring; a boy soprano can suddenly hardly carry a tune. It has not been the ethical standard of care to stop puberty so that these changes can be circumvented.

43. Erikson described the stage of adolescence as "Identity versus Role Confusion" during which the teen works at developing a sense of self by testing roles then integrating them into a single identity.[23] This process is often unpleasant regardless of the presence or absence of gender identity conflicts. The major benefit of enduring puberty in a GD patient is that it provides a strong likelihood of alignment of his gender identity with his natal sex. There is no doubt that these patients need compassionate care to get them through their innate pubertal changes. The light at the end of the tunnel for them is the proven scientific evidence that 80%-95% of pre-pubertal children with GD will come to identify with their biological sex by late adolescence. Some will require lifelong supportive counseling, and others will not.[24]

44. Intervention at a young age with gonadotropin releasing hormone analogs (often referred to as puberty blockers) to either stop puberty early on or prevent it from starting before it naturally occurs is suggested by guidelines developed by WPATH without scientific basis.

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[23] Erikson, E. H. (1993). *Childhood and society*. WW Norton & Company. Erikson, E. H. (1993). *Childhood and society*. WW Norton & Company.

[24] Zucker KJ, Gender Identity Disorder, in Rutter M, Taylor EA, editors. *Child and Adolescent psychiatry*, 4th ed, Malden Mass: Blackwell, 2006: 737-753.

There is evidence that bone mineral density is irreversibly decreased if puberty blockers are used during the years of adolescence.[25]

45. To treat puberty as a pathologic state of health that should be avoided by using puberty blockers (GnRH analogs) is to interrupt a major necessary physiologic transformation at a critical age when such changes can effectively happen. We have definite evidence of the need for estrogen in females to store calcium in their skeleton in their teen years. That physiologic event can't be put off successfully to a later date. It is very difficult to imagine ethical controlled clinical trials that could elucidate the effects of delaying puberty until the age of consent (18 years). The use of cross-sex hormones during this same time frame has no basis of safety and efficacy. The use of such treatment in adults raises scientifically valid concerns that were amply expressed in the 2009 Endocrine Society Guidelines on Transgender treatment.

46. The next step in WPATH-recommended intervention is to use cross-sex hormone therapy during the time when the patient would naturally be experiencing endogenous pubertal changes. This too is not based on scientifically proven theories. The use of cross-sex hormones can cause permanent infertility.[26]

47. The final recommended step is so-called "sex reassignment surgery," which can include surgical removal of the breasts in natal females, or removal of the penis and scrotum in natal males. Each of these steps have adverse outcomes, some reversible and others not. Mastectomies leave scars, and there is great difficulty in creating a functional vaginal-like orifice, and certainly no success in creating an innervated erectile penis where none existed previously. Sex reassignment surgery is, by nature, permanent.

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[25] *J Clin Endo Metab* 2008;93:190-195.

[26] Hembree WC et al, Endocrine Treatment of Transsexual Persons: and Endocrine Society Clinical Practice Guideline, *J Clin Endo Metab*2009; 94:3132-3154.

Science vs. Pseudoscience

48. The advent of "centers of excellence" for gender-disordered patients[27] combined with sociologic agenda in academia has created the impression that there is scientific validity to gender discordance as a variation of normal. There has been a flurry of non-peer-reviewed articles in journals and newsletters circulated to general pediatricians that promote the ideology of transgenderism without scientific support.[28, 29, 30, 31] Mainstream clinicians and scientists who consider gender discordance to be a mental disorder have been deliberately excluded in the makeup of the steering committees of academic and medical professional societies which are promulgating guidelines that were previously unheard of.

49. The Endocrine Society published such a document in 2009.[32] Its recommendations promoted the use of psychological evaluation, counseling, blocking of pubertal maturation at the onset of puberty, the subsequent use of cross- sex hormones, and possible surgical intervention at the age of consent. Of the 22 recommendations contained in the document, only three were supported by scientific proof. These three warned of potential adverse effects of hormonal manipulation. The remaining 19 recommendations were nearly evenly split into a group that was based on very limited scientific evidence and a group that was based on absolutely no scientific evidence at all. The response to these guidelines was an exponential burgeoning of Gender Identity Clinics in the United States from three to over forty-five in a period of seven years.

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[27] Hsieh S and Leninger J, Resource List: Clinical Care Programs for Gender-Nonconforming Children and Adolescents, *Pediatr Ann* 2014:43:238-244.
[28] Prager, LM, A boy who wants to be a girl, *Contemporary Pediatrics 2008*; 25:56-58.
[29] Garafolo R Tipping points in caring for the gender-non-conforming child and adolescent, *Pediatr Ann* 2014; 43:227-229.
[30] Steever J, Cross-gender Hormone therapy in adolescents, *Pediatr Ann* 2014;43: e-138-e-144.
[31] Simons LK et al, Understanding gender variance in Children and Adolescents, *Pediatr Ann*2014;43:e-126-e131.
[32] Hembree WC et al, Endocrine Treatment of Transsexual Persons: and Endocrine Society Clinical Practice Guideline, *J Clin Endo Metab*2009; 94:3132-3154.

50. What is missing is sound science to show that gender identity discordance is not a delusional state. What is happening is reminiscent of the now-discredited efforts of John Money. There is an ongoing multicenter study, funded by the NIH, which will be empirically starting gender discordant children and adolescents, all below the age of consent, on treatment with puberty blockers and cross-sex hormone treatments. There is no control group in this study.

51. The gender discordant individual is given protected civil rights as if the discordant gender identity is innate, when there is no credible science to prove such, and in fact, much credible science to refute it. Recognized experts in the field, such as Kenneth Zucker, are banned from providing psychotherapy to assist youth in aligning their gender identity with their biological sex.

52. The norm for human development is for one's thoughts to align with physical reality, and for one's gender identity to align with one's biologic sex. Gender identity that does not match natal sex is a mental disorder, previously called Gender Identity Disorder.

53. WPATH is an agenda-driven advocacy organization whose membership consists of anyone who has an interest in the transgender social and political agenda. There are no requirements for specialty training or certification. Its guidelines are not scientifically supported.

54. WPATH promotes "expert witnesses" and provides them with a bibliography replete with self-confirming references to opinion pieces and anecdotal case reports along with clinical case reviews with inherent selection bias.

55. WPATH's "peer-reviewed" journal is not reviewed by anyone with an opinion that is not in keeping with the philosophy of the organization itself. WPATH pressured the authors of the Swedish study to retract their results clearly showing persistent mental health problems among transgendered adults even after WPATH-recommended transition treatment. When Dr. Zucker,

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the then-editor of the journal that published the study, refused to allow the authors do so, he was targeted by activists and his Toronto clinic was shut down by the Canadian government shortly thereafter.

56. Laws banning treatment designed to support gender resolution appear aimed at a fear that allowing the gender discordant individual to return to their chromosomal sexual identity will do harm, when what we know is that he or she will most likely assume a role as a heterosexual or homosexual adult living in and identifying with the body given them by nature.

Conclusion

57. Young children and adolescents are vulnerable to recruitment to an ideology of gender fluidity, which is theorized by various agenda-driven health professionals and groups, and which is amplified on the internet by profoundly unscientific websites and blogs. After my fellowship completion, it was not until 16 years later that I encountered a patient with Gender Identity Disorder. At that time, I consulted all of the mentors in pediatric endocrinology across the country that I respected, and none of them could give me a suggestion of where to send the patient for valid psychological care. Since the flurry of published articles lacking in valid science, and the emergence of transgender clinics across the nation in recent years, I have seen an uptick in case referrals. Based on the proven results of Kenneth Zucker, I seek out and send these patients to competent mental health providers who thoroughly assess the family psychological environment, treat any psychological comorbidities in the child, and support the child through puberty. This course of treatment offers patients the best hope of recovery and a healthy, productive life.

58. Pursuant to 28 U.S.C § 1746, I declare under penalty of perjury under the laws of the United States of America that the foregoing is true and correct.

Date: 12 August, 2016

Signed: 

Quentin L. Van Meter, M.D.

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PERSONAL

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EDUCATION:

Undergraduate:	College of William & Mary, 1969 B.S. – 1969
Medical School:	Medical College of Virginia, 1973 M.D. – 1973

CLINICAL TRAINING:

Institution:	The University of California, San Francisco
Hospital:	Naval Regional Medical Center, Oakland
Position:	Pediatric Intern – 1973 – 1974 Pediatric Resident – 1974 – 1976
Institution:	Johns Hopkins University
Hospital:	Johns Hopkins Hospital
Position:	Fellow, Pediatric Endocrinology 1978 – 1980 Fellowship Program Director: Claude Migeon, M.D.
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PROFESSIONAL CERTIFICATION & SOCIETIES:

Diplomate, National Board of Medical Examiners, 1974

American Board of Pediatrics, certified in general pediatrics, 1978, sub-board certified in Pediatric Endocrinology, 1983

Fellow:	American Academy of Pediatrics, 1975 -2014 President, Uniformed Services West Chapter, 1987 – 1990 District VIII member, AAP Committee on Awards for Excellence in Research, 1990-1994 Editor, The Georgia Pediatrician, 1994 – 1998
	Chairman, Georgia Chapter Legislative Committee, 1996 – 2006
Fellow:	The American College of Pediatricians, 2007 – present Member of the Board of Directors, 2008- present Vice President/President Elect, 2015-present
Member:	Pediatric Endocrine Society, 1989 – present
Member:	American Diabetes Association Professional Section, 1988 – present
Member:	Endocrine Society, 1994-present
Member:	Southern Pediatric Endocrine Society, 1992 – Present
Member:	American Association of Clinical Endocrinologists, 2005 – present
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FACULTY POSITIONS:

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Institution:	Emory University School of Medicine
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Institution:	University of California, San Francisco
Position:	Associate Clinical Professor, Pediatrics, 1989 – 1991
Institution:	University of California, San Diego, School of Medicine
Position:	Assistant Clinical Professor, Pediatrics, 1980 – 1986
Institution:	LSU School of Medicine, Clinical Instructor, Pediatrics, 1977 – 1978

MILITARY SERVICE:

Commission:	Medical Corps, United States Navy, August 1971
Rank:	Captain, retired
Duty Stations:	Health Professional Scholarship Student, 1971 – 1974
	Intern and Resident, Pediatrics, Naval Regional Medical Center, Oakland, 1973 – 1976
	Staff Pediatrician, Naval Regional Medical Center, Oakland, 1976

Staff Pediatrician, Naval Regional Medical Center,
New Orleans, 1976 – 1978

Full time out-service fellow in Pediatric Endocrinology,
Johns Hopkins Hospital, 1978 – 1980

Staff Pediatric Endocrinologist, Naval Hospital San Diego,
1980 – 1986

Chairman and Director, Residency Training, Department of Pediatrics
Naval Hospital Oakland, 1986 – 1991

OTHER PROFESSIONAL ACTIVITIES:

Consultant, Pediatric Endocrinology,
Nellis Air Force Base Hospital, Las Vegas, Nevada
1981 – 1991

Consultant, Pediatric Endocrinology,
Naval Hospital Lemoore, CA
1986 – 1991

Consultant, Pediatric Endocrinology,
Letterman Army Medical Center, Presidio of San Francisco, CA
1990 – 1991

Consulting Endocrinologist,
Columbus Regional Medical Center, Columbus, GA
1991 – 1994

Pediatrician and Pediatric Endocrinologist, partner
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Speaker's Bureau
Novo Nordisk, Pfizer, Endo, Abbvie
AAP Eqipp course on Growth- development committee- 2012

PUBLICATIONS: (Articles in Peer Reviewed Journals)

Riddick, JR, Flora R., Van Meter, QL:
"Computerized Preparation of Two-Way Analysis of Variance Control Charts for Clinical Chemistry," Clinical Chemistry, 18:250, March 1972.

Van Meter, QL, Gareis FJ, Hayes, JW, Wilson, CB:
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Plotnick, LP, Van Meter, QL, Kowarski, AA, "Human Growth Hormone Treatment of Children with Growth Failure and Normal Growth Hormone Levels by Immunoassay: Lack of Correlation with Somatomedin Generation: Pediatrics 71:324, March 1983.

Brawley, RW, Van Meter, QL, "Mebendazole Ascaris Migration," W.J. Med, 145:514015, October 1986.

Van Meter, QL, "The Role of the Primary Care Physician in Caring for Patients with Type-1 Diabetes," Comp Ther 1998; 24(2):93–101

Midyett LK, Rogol AD, Van Meter QL, Frane J, and Bright GM, "Recombinant Insulin-Like Growth factor (IGF)-I Treatment in Short Children with Low IGF-I Levels: First-Year Results from a Randomized Clinical Trial," J Clin Endocrinol Metab, 2010;95:611–619.

ABSTRACTS:

Van Meter,Q L, & Lee, PA: "Evaluation of Puberty in Male and Female Patients with Noonan Syndrome," Pediatric Research 14:485, 1980.

Van Meter, QL, et al: "Characterization of Pituitary Function in Double Bolus GnRH Infusion as a Diagnostic Tool," Pediatric Research 32:111, 1984.

Van Meter, QL, Felix, SD, Lin, FL: "Evaluation of the Pituitary-Adrenal Axis in Patients Treated with nasal Beclomethasone," (Presented at the 1991 Annual Meeting of the Endocrine Society and the 6th Annual Naval Academic Research Competition, Bethesda, MD, 17 May, 1991).

Rogol AD Midyett LK Van Meter Q, Frane J, Baily J, and Bright GM, Recombinant Human IGF-1 for Children with Primary IGF-1 Deficiency (IGFD): Safety Data from Ongoing Clinical Trials (presented at the PAS 2007, Toronto).

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Van Meter Q, Midyett LK, Deeb L et al, Prevalence of primary IGFD among untreated children with short stature in a prospective, multicenter study (Poster POO715) ICE Rio de Janeiro, Brazil 2008.

G.M. Bright[1], W.V.Moore[2], J.Nguyen[3], G. Kletter[4], B. S. Miller[5], Q. L. Van Meter[6], E. Humphriss[1], J.A. Moore[7] and J.L. Cleland[1] Results of a Phase 1b Study of a new long-acting human growth hormone (VRS-317) in pediatric growth hormone deficiency (PGHD). PAS 2014 May 2014

Van Meter Q, Welstead B and Low J, Characteristics of a Population of Obese Children and Adolescents: Suggesting a New Paradigm, presented at ESPE meeting, Dublin 2014.

ADDITIONAL PRESENTATIONS/LECTURES:

Pediatrics Update, CME Associates, San Diego – Orlando Annual Conferences: Lectures on Pediatric Endocrine Subjects – 1986 – 2001. Course Moderator, 1997, 1998, 1999, 2000, 2001

Endocrine and Gastroenterology Update, CME Associates, Maui HI Nov 2001, Lecturer and Course Moderator

Lecture on Panhypopitutarism, Pharmacia Conference, Nashville TN April 2002.

Family Medicine Review Course, Orlando, FL, 1992 – 2001

Pediatric Grand Rounds, Tanner Medical Center, October 1997

Pediatric Grand Rounds, Hughes Spaulding Children's Hospital, September, 2003

Pediatrics in the Park, Fall CME meeting for the Georgia Chapter of the American Academy of Pediatrics, November 2003

Pediatric Grand Rounds, Columbus Regional Medical Center, January 2004

Frontiers in Pediatrics CME Course, sponsored by the Atlanta Children's Health Network, Atlanta, March 2004.

Pediatric Grand Rounds, Eggleston Children's Hospital, May 2004.

Sue Schley Matthews Pediatric Conference, Columbus Regional Medical Center, September 2004

56th Annual Scientific Assembly and Exhibition of the Georgia Academy of Family Physicians, Nov 2004

Program Co-Chairman: Southern Pediatric Endocrine Society Annual meeting, Nov 2004

Presentations on Diabetes, Growth Failure, and Thyroid Disease to the Postgraduate Pediatric Nurse Practitioner Program, Georgia State University, Nov 2005, June 2006, May 2007

Issues in Medicine, US Medical Congress Conference and Exhibition, Las Vegas, meeting planner and speaker, June, 2006

CME Presentations for the Georgia Chapter of the American Academy of Pediatrics Spring and Fall Meetings 2004-present

Pediatric Grand Rounds, Columbus Regional Medical Center, Columbus, GA, 2011-present

Human Growth Foundation Regional CME Conference, Atlanta GA March 2013, February 2014 Columbus Georgia

Audio Digest Pediatrics - ① v. 41, no. 4; ② v. 41, no. 20; ③ v. 43, no. 17

Audio Digest Family Practice - ① v. 42, no. 5; ② v. 44, no. 11; ③ v. 44, no. 44; ④ v. 45, no 15

Audio Digest Otolaryngology - ① v. 32, no. 14

CURRENT HOSPITAL APPOINTMENTS:

Eggleston/Scottish Rite Children's Hospitals, active staff, Pediatric Endocrinology

PAST AND CURRENT CLINICAL RESEARCH:

2006	Sanofi-Aventis HMR1964D/3001	study completed 2007
2006	Tercica MS301-	study completed 2008
2007	Tercica MS310-	study completed 2008
2007	Tercica MS306-	study completed 2010
2007	Tercica MS316-	study completed 2012
2008	EMD Serono 28358	study completed 2009
2012	Versartis 12VR2	study completed 2014
2012	Debiopharm 8206-CPP-301	study started July 2012
2013	Versartis 13 VR3	study started Dec 2013
2014	Novo-Nordisk Elipse	study started 2014
2015	Versartis 14 VR4	study started September 2015